POSTTRAUMATIC STRESS: AN INFORMATION PACKET

NATIONAL CENTER FOR POST-TRAUMATIC STRESS DISORDER

∴ BOSTON ∴ PALO ALTO/MENLO PARK ∴
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POSTTRAUMATIC STRESS INFORMATION PACKET

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POSTTRAUMATIC STRESS

When a terrifying, uncontrollable event overwhelms a person's sense of safety and security, a normal and predictable reaction to the event occurs. Traumatic stress can result from very different life threatening events including natural disaster, sexual abuse/assault, war zone duty, or the witnessing of terrible things happening to other people. Accidental disasters (i.e. car, train, boat, airplane, fires, explosions); natural disasters (i.e. floods, tornadoes, hurricanes, earthquakes), or deliberately caused disasters (i.e. bombings, shootings, torture, rape, assault and battery) are all capable of producing stress. Although there are many types of traumatic events, survivors experience a common stress response which may include feelings of terror, vulnerability, helplessness, fear of bodily injury, or overwhelming loss and guilt over actions taken or avoided. The typical pattern of response to various forms of traumatic events is recognized as a *posttraumatic stress reaction or syndrome.*

Posttraumatic stress syndrome is a normal reaction to an abnormal event. It can occur at any age, including childhood. Most survivors of trauma recover to the extent that they are symptom free, however, they often remain vulnerable to stimuli directly reminiscent of the original trauma. The impact of trauma upon the survivor (referred to as primary traumatization) generally begins immediately or soon after the event, though delayed reactions have been observed. The impact may be mild or severe, sometimes affecting nearly every aspect of the survivor's life. The impact may even affect other significant people in the survivor's life (referred to as secondary traumatization). The survivor may experience sleep disturbance, emotional instability, impaired concentration, and increased stress within relationships. The abuse of alcohol or other psychoactive substances is common, particularly if the survivor is predisposed to substance abuse.

A small percentage of victims develop chronic symptoms of post-traumatic stress. These individuals appear to develop a generalized sensitivity to stimuli not directly related to the original trauma, which can be observed in the activation of symptoms by seemingly unrelated events. For these survivors, the posttraumatic stress syndrome has developed into *Posttraumatic Stress Disorder (PTSD),* requiring psychosocial and/or psychiatric treatment to rduce the symptoms and the disturbance of their social life/relationships, identity, and day-to-day functioning.

Although *The Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) is used for the assessment of pathological syndromes, the conceptual framework the manual uses for determining post-traumatic stress disorder is also useful in conceptualizing post-traumatic stress syndrome. Similar to post-traumatic stress disorder, normal post-traumatic stress is characterized by three major response patterns: a *re-experiencing* of the event, *behavioral avoidance and/or emotional numbing* of stimuli associated with the event, and increased *physiological arousal.*

POSTTRAUMATIC STRESS SYNDROME: CARDINAL FEATURES

REEXPERIENCING

BEHAVIORAL AVOIDANCE/ PSYCHIC NUMBING

INCREASED PHYSIOLOGICAL AROUSAL

Reexperiencing

Very often a survivor may experience a heightened sensitivity to stimuli reminiscent of the original trauma resulting in their reexperiencing (in thought, emotion, behavior, or physiology) the original event. For example, earthquake survivors may become disturbed when a passing truck causes their home to vibrate; the sound of rain may upset flood survivors; the sound of a helicopter may cause discomfort in a Vietnam veteran. In each case, the survivor may respond as if the original event were reoccurring. Intrusive recollections of the event is another form of reexperiencing. Flood survivors may involuntarily think about the images of rushing water through their home, vehicular accident survivors may involuntarily visualize the scene of the crash.

Behavioral Avoidance and or Emotional Numbing

Survivors often attempt to avoid thoughts or feelings about the traumatic event or activities that evoke memories of it. The blunting of feeling may result in indiscriminate psychic numbing or psychogenic amnesia. For instance, a survivor of incest may describe events in a way that is devoid of any affect, or a victim of rape may not remember what the attacker looked like. Victims of home burglaries may refuse to enter their homes alone; natural disaster survivors may refuse to engage in disaster preparedness.

Arousal

Often the survivor experiences symptoms of increased physiological arousal. The heightened sensitivity to stimuli reminiscent of the original trauma may result in hypervigilance, exaggerated startle response, and/or an increase in blood pressure, pulse rate, and other physiologic reactivity. This

increased arousal may cause sleep disturbance, impaired concentration, and increased irritability.

Another way of organizing the characteristics of post-traumatic stress is by separating symptoms into four response categories: *emotional, cognitive, biological, and psychosocial* (Table 1).

TABLE 1

COMMON RESPONSES TO A TRAUMATIC EVENT

EMOTIONAL	COGNITIVE
SHOCK	IMPAIRED CONCENTRATION
DISBELIEF	CONFUSION
ANGER	SELF-BLAME
RAGE	DISORIENTATION
TERROR	DECREASED SELF-ESTEEM/EFFICACY
GUILT	INTRUSIVE THOUGHTS
GRIEF	
VULNERABILITY	
IRRITABILITY	
HELPLESSNESS	

BIOLOGICAL	PSYCHOSOCIAL
FATIGUE	ALIENATION
INSOMNIA	SOCIAL WITHDRAWAL
NIGHTMARES	INCR. STRESS WITHIN RELATIONSHIPS
HYPERAROUSAL	SUBSTANCE ABUSE
STARTLE RESPONSE	VOCATIONAL IMPAIRMENT

POSTTRAUMATIC STRESS DISORDER

Posttraumatic stress disorder (PTSD) is a *prolonged* post-traumatic stress response. In addition, there may be much greater personality and social impairment than evidenced in post-traumatic stress syndrome as an individual accommodates to the presence of these symptoms over a long period of time. Although there are several factors that affect a victim's response, generally, survivors who experience severe, frequent, or extended sensory exposure to a traumatic stressor(s), are at higher risk for developing chronic symptoms. The same characteristics of post-traumatic stress apply to PTSD; however, the symptoms will have greater intensity, higher frequency, and longer duration.

The *DSM-IV* criteria for PTSD (Table 2) require a minimum set of symptoms: *one* symptomatic form of *re-experiencing* the traumatic event, a minimum of *three* symptoms of persistent *avoidance* of stimuli associated with the trauma, and a minimum of two persistent symptoms of increased *arousal*. The *duration* of the disturbance (symptoms in B, C, and D) according to *DSM-IV* must be at least one month. In addition, a new criterion, (F), was added to include *clinically significant distress or impairment in social, occupational, or other important areas of functioning*. If the duration of symptoms is less than 3 months, the disorder is categorized as *Acute*; if symptoms persist for 3 months or more, the disorder is categorized as *Chronic*. If onset of symptoms is at least 6 months after the stressor, it is categorized *With Delayed Onset*.

TABLE 2

PTSD: CARDINAL FEATURES

REEXPERIENCING
BEHAVIORAL AVOIDANCE/ PSYCHIC NUMBING
INCREASED PHYSIOLOGICAL AROUSAL
DURATION (AT LEAST 30 DAYS)
CLINICALLY SIGNIFICANT DISTRESS OR IMPAIRMENT IN SOCIAL, OCCUPATIONAL, OR OTHER IMPORTANT AREAS OF FUNCTIONING

DIAGNOSTIC CRITERIA FOR POSTTRAUMATIC STRESS DISORDER (DSM-IV)

A . The person has been exposed to a traumatic event in which both of the following were present:

(1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others

(2) the person's response involved intense fears, helplessness, or horror.
Note: In children, this may be expressed instead by disorganized or agitated behavior

B. The traumatic event is persistently reexperienced in one (or more) of the following ways:

(1) recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. **Note:** In young children, repetitive play ma occur in which themes or aspects of the trauma are expressed.

(2) recurrent distressing dreams of the event. **Note:** In children, there may be frightening dreams without recognizable content.

(3) acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated). **Note:** In young children, trauma-specific reenactment may occur.

4) intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:

(1) efforts to avoid thoughts, feelings, or conversations associated with the trauma

(2) efforts to avoid activities, places, or people that arouse recollections of the trauma

(3) inability to recall an important aspect of the trauma

(4) markedly diminished interest or participation in significant activities

(5) feeling of detachment or estrangement from others

(6) restricted range of affect (e.g., unable to have loving feelings)

(7) sense of a foreshortened future (e.g., does not expect to have a career, marriage, or children, or a normal life span)

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by at least two (or more) of the following:

(1) difficulty falling or staying asleep

(2) irritability or outbursts of anger

(3) difficulty concentrating

(4) hypervigilance

(5) exaggerated startle response

E. Duration of the disturbance (symptoms in Criteria B, C and D, is more than 1 month).

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

ACUTE STRESS DISORDER

The *DSM-IV* (1994) introduced a new category of Posttraumatic Stress Disorder: Acute Stress Disorder. The essential feature of this stress disorder is the development of anxiety, dissociative, and other symptoms that occur within 1 month of exposure to a traumatic stressor. Acute Stress Disorder is characterized by five major response patterns: *dissociation or emotional numbing of stimuli associated with the event, a re-experiencing of the event, behavioral avoidance, increased physiologic arousal, and social-occupational impairment.* To meet the diagnostic cut off, a person must exhibit *three* or more of the *dissociative symptoms*, and *at least one* form of *re-experiencing, behavioral avoidance, physiologic arousal, and significant social and or occupational impairment.* The disturbance must last for a minimum of 2 days and a maximum of weeks within four weeks of the traumatic event.

Dissociation/emotional numbing

Survivor's may experience indiscriminate dissociation, detachment, or absence of emotional responsiveness; a reduction in awareness of his or her surrounding; derealization; depersonalization; or dissociative amnesia. For example, a survivor of incest may describe events in a way that is devoid of any affect, or a victim of rape may not remember what the attacker looked like and feel detached from her body, or a disaster survivor may experience the world, other people, and him or her self as unreal and dreamlike.

Re-experiencing

Very often a survivor may experience a heightened sensitivity to stimuli reminiscent of the original trauma resulting in their reexperiencing (in thought, emotion, behavior, or physiology) the original event. For example, earthquake survivors may become disturbed when a passing truck causes their home to vibrate; the sound of rain may upset flood survivors; the sound of a helicopter may cause discomfort in a Vietnam veteran. In each case, the survivor may respond as if the original event were reoccurring. Intrusive recollections of the event is another form of re-experiencing. Flood survivors may involuntarily think about the images of rushing water through their home, vehicular accident survivors may involuntarily visualize the scene of the crash.

Behavioral avoidance

Stimuli associated with the trauma are persistently avoided. The person commonly makes deliberated efforts to avoid thoughts, feelings or conversations about the traumatic event. Victims of home burglaries may refuse to enter their homes alone; natural disaster survivors may refuse to engage in disaster preparedness.

Arousal

Often the survivor experiences symptoms of increased physiological arousal. The heightened sensitivity to stimuli reminiscent of the original trauma may result in hyper vigilance, exaggerated startle response, and/or an increase in blood pressure, pulse rate, and other physiologic reactivity. This increased arousal may cause sleep disturbance, impaired concentration, and increased irritability.

DIAGNOSTIC CRITERIA FOR ACUTE STRESS DISORDER (DSM-IV)

A. The person has been exposed to a traumatic event in which both of the following were present:

(1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others

(2) the person's response involved intense fears, helplessness, or horror.

B. Either while experiencing or after experiencing the distressing event, the individual has three (or more) of the following dissociative symptoms:

(1) a subjective sense of numbing, detachment, or absence of emotional responsiveness

(2) a reduction in awareness of his her surrounding (e.g., "being in a daze")

(3) derealization

(4) depersonalization

(5) dissociative amnesia (i.e., inability to recall an important aspect of the trauma)

C. The traumatic event is persistently reexperienced in at least one of the following ways: recurrent images, thoughts, dreams, illusions, flashback episodes, or a sense of reliving the experience, or distress on exposure to reminders of the traumatic event.

D. Marked avoidance of stimuli that arouse recollections of the trauma (e.g., thoughts feelings, conversations, activities, places, people).

E. Marked symptoms of anxiety or increased arousal (e.g. difficulty sleeping, irritability, poor concentration, hypervigilance, exaggerated startle response, motor restlessness).

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning, or impairs the individual's ability to pursue some necessary task, such as obtaining necessary assistance or mobilizing personal resources by telling family members about the traumatic experience.

G. The disturbance lasts for a minimum of 2 days and a maximum of 4 weeks and occurs within 4 weeks of the traumatic event.

H. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition, is not better accounted for by Brief Psychotic Disorder, and is not merely an exacerbation of preexisting Axis I or Axis II disorder.

POSTTRAUMATIC STRESS
ASSESSMENT AND TREATMENT: AN OVERVIEW

Assessment

Several factors may affect the intensity and duration of a survivor's post-traumatic stress response. These include the degree of sensory exposure to the traumatic stressor(s), personality characteristics, and pre-trauma adjustment and functioning. Additional factors include the availability and utilization of support systems, and the characteristics of the post-traumatic environment.

It is essential that the assessment of an individual's adjustment to post-traumatic stress be made in the context of the survivor's psychosocial history. A list of questions to clarify important psychosocial issues are presented in Table 3.

TABLE 3

PSYCHOSOCIAL ISSUES

* What other significant events or previous losses have occurred in the survivor's life prior to the traumatic event?
* How did the survivor adapt to these events?
* What current life stressors was the survivor experiencing at the time of the traumatic event?
* How much social support does the survivor have? Is the survivor accessing social support?
* What does the event mean to the survivor?
* How has the event affected the survivor's social life, job, health?
* What coping strategies does the survivor use?
* Does the survivor have a history of substance abuse?

Information gained from such questions will provide an indication of the traumatic event's impact and a direction for providing support to the survivor, be it in the form of referral, counseling, or in-depth assessment/treatment.

There are several diagnostic instruments used to assess PTSD. A partial list is presented in Table 4.

TABLE 4

PTSD ASSESSMENT INSTRUMENTS

Diagnostic Instruments
Structured Clinical Interview for DSMIII-R (SCID)
Clinician-Administered PTSD Scale (CAPS)
Psychological Tests /Measures
Minnesota Multiphasic Personality Inventory (MMPI)
Mississippi Scale for Combat Related PTSD
Civilian Mississippi Scale for PTSD
Impact of Event Scale (IES)

In addition, psychophysiological protocols (Keane, et al., 1988; Pitman, et al., 1987) and neurobiological assessment techniques (Mason, et al., 1990) are being developed. Projective tests are sometimes useful in the assessment of PTSD (e.g., Rorschach and drawings).

Treatment

Numerous treatments have been utilized to assist the adjustment process of traumatized individuals. It is beyond the scope of this manual to review the variety of treatments; however, exposure-based behavioral treatments (flooding and systematic desensitization), cognitive-behavioral treatments, and pharmacotherapy have shown considerable promise in the amelioration of symptoms associated with PTSD. In addition, ancillary techniques of stress management and expressive art therapies have been used successfully. Common treatment approaches are listed in Table 5.

TABLE 5

COMMON PTSD TREATMENT APPROACHES

Individual psychodynamic psychotherapy
Individual behavior therapy
Individual cognitive-behavior therapy
Group psychotherapy
Marital and family therapy
Pharmacotherapy
Abreactive treatments
Multi-modal treatment integration

Treatment modalities must be selected in view of specific treatment objectives. For example, cognitive treatments may ameliorate irrational styles of thinking, while medications may serve to reduce the physiologic reactivity of the trauma response. A group approach may provide the opportunity to enhance trust, decrease the tendency to isolate, reduce stigmatization and thus increase the social support of the victim. The multidimensional characteristics of post-traumatic stress disorder necessitate identifying the appropriate and optimal patient-treatment match.

Treatment of PTSD/Alcohol Co-morbidity

In 1988, the Research Triangle Institute published the results of the most rigorous epidemiological study of combat soldiers ever conducted. Among other findings, the National Vietnam Veterans Readjustment Study (NVVRS) found a lifetime prevalence of PTSD in combat veterans to be over thirty percent (30%). At one time or another, seventy-five (75%) of these veterans, have also met the criteria for alcohol abuse or dependence. Among female Vietnam veterans, having a current diagnosis of PTSD predicts a greater than fivefold increase in the likelihood that alcohol abuse or dependence is a current problem. Nearly half of all veterans who have ever suffered from PTSD and alcoholism still experience this dual disorder today.

Regardless of your setting, be it an inpatient unit, outpatient clinic, or a Vet Center, you will encounter veterans with symptoms of PTSD and alcohol dependence/abuse. According to NVVRS findings, male veterans with PTSD are much more likely to abuse alcohol or drugs than veterans who do not have PTSD. Further findings indicated that veterans who meet the criteria for either alcohol abuse or dependence suffer more impairment on almost every measure of post-war adjustment and psychological well-being than their cohorts who don't meet the criteria. Dually symptomatic veterans are less likely to have completed high school, less likely to be employed and/or have occupational stability, and are less likely to be married. They are more likely to have had multiple divorces and report greater parental difficulties. They are more socially isolated, more prone to violence, and not surprisingly, report low levels of well-being.

When a veteran with PTSD has a co-morbidity of alcohol abuse, should he be treated in the substance abuse program or the post-traumatic stress clinic? It is not uncommon for clinicians to ask "What is the most effective treatment sequence for the dually disordered patient?" The question of which disorder to treat first, is the wrong question. The correct question is how to treat both problems simultaneously. Treatment of PTSD/Alcohol Dependence co-morbidity requires that the patient be substance free with an emphasis placed on the effort to understand the veteran as a whole person. In other words, it more important to recognize the interactive factors of a veteran's developmental and traumatic history, the veteran's biological disposition, and beliefs about himself and alcohol, than it is to solely isolate and target a symptom of PTSD or alcohol abuse for treatment.

The physiologic reactivity associated with PTSD is distressful in and of itself, and may trigger other painful symptoms as well. To suppress the symptoms of hyperarousal, PTSD patients often resort to alcohol. However, the emotional and behavioral dysinhibition that occurs with intoxication is terrifying to most veterans with PTSD. A rebound pattern develops and is eventually established at great cost, as this pattern results in the exacerbation of symptoms of both disorders. Disrupted families, criminal behavior, ruined careers, impaired physical health, social isolation, suicidal behavior, and untimely death are the tragic effects.

The general literature implies that successful treatment of concurrent PTSD and substance abuse may require prompt control of PTSD symptoms, combined with simultaneous substance abuse treatment (Kofoed, Friedman, & Peck, in press).

Although there are several treatment models that address PTSD and several treatment models that address alcohol abuse, hybrid treatment models are beginning to appear. The effort to blend and integrate PTSD and alcohol treatment strategies have been based on theoretical models, clinical experience, and clinical judgment, however, there is little outcome data to differentiate the efficacy of any specific approach.

Three treatment goals are generally considered when treating the dually diagnosed veteran (Abueg & Fairbank, 1991). One treatment goal is to reduce the physiologic reactivity and re-experiencing symptoms associated with PTSD. A second goal is to assist the veteran toward developing coping behaviors that counteract the maladaptive coping style associated with PTSD and alcohol abuse (i.e., social withdrawal, hostility, helplessness, etc.). A third general goal is reducing addictive behaviors.

The complex mix of symptoms and interwoven relapse risk factors of the combined disorders require that any treatment plan be individualized. Abueg and Fairbank (1991) have described a sequential treatment approach that includes building and sustaining motivation for treatment, decreasing active PTSD symptoms, and building more effective social and problem solving skills. Specific individualized behavioral and cognitive strategies are developed for relapse prevention, utilizing group therapy, direct therapeutic exposure, and cognitive problem solving training.

Other clinicians (Bollerud, 1990; Jellinek, 1984; Schnitt & Nocks, 1984) have attempted to integrate group and individual therapies of PTSD with the components of "Twelve Step" programs, i.e., Alcoholics Anonymous. Using the dynamics of group support and confrontation along with individual psychotherapy of PTSD, these treatment approaches look to nurture motivation and interpersonal rewards for an abstinent lifestyle, while simultaneously aiming to help the patient validate and integrate the emotional and cognitive experiences of the trauma. Regardless of the model adopted, it is necessary that the clinician, the veteran and the significant people in his life, are in agreement that the veteran has a disorder requiring long-term treatment.

Services for the dually diagnosed veteran can incorporate the components cited in Table 6 and Table 7 into an integrated program in either outpatient or inpatient setting. Although no research exists regarding the superiority of either outpatient or inpatient setting in PTSD treatment, the assessment of severity, danger to self/others, are primary indicators for disposition.

TABLE 6

CO-MORBIDITY TREATMENT ISSUES OF PTSD/ALCOHOL ABUSE DEPENDENCE

physical well being	affect management
problem solving/coping styles	social relatedness/ support systems
cognitive analysis of content, context and meaning of combat experience	identifying high risk situations likely to lead to relapse
social skills development	maintaining sobriety

TABLE 7

CO-MORBIDITY TREATMENT INTERVENTIONS FOR PTSD/ALCOHOL ABUSE DEPENDENCE

* Medical/Psychological Aspects of Substance Abuse
* Growing up with Alcoholism (patient's family of origin and effects upon patient)
* Trauma Focus (combat group with special attention to alcohol use in Vietnam)
* Family Therapy (attending to co-dependency)
* Stress Management/Relaxation
* Relapse Prevention Training

Group treatment is an appropriate intervention for discussing and treating issues that are common to both PTSD and alcohol abuse/dependence. Issues such as low self-esteem, guilt, anxiety, lack of trust, social deviance, and moral failure can be examined within the context of therapeutic and peer support.

As mentioned earlier, treating a veteran with a dual diagnosis requires examining the veteran's home environment, including the veteran's family (family not necessarily being limited to the traditional family). An estimated one million children are victims of secondary traumatization as a result of repeated exposure to a parent with chronic PTSD. These children can suffer from emotional and physical abuse. In addition, they are subjected to the complication of living with someone who has a substance abuse problem. Consequently, family therapy is an important component of treatment. It

may be that the entire family will need to learn new ways to solve old problems, including developing effective communication skills, negotiation, decision-making, and conflict resolution.

As a clinician, be aware that a patient's symptoms may worsen as treatment progresses and the patient's awareness increases. More impulsive patients will often direct anger toward family, clinical staff, or other patients. As the patient's awareness intensifies, so may the tendency to seek relief from alcohol or other substance abuse. Your awareness of this pattern and your reassurance of the patient in a firm, limit-setting context can diffuse the patient's anger and discomfort.

Medications must be prescribed cautiously in view of the patient's history of substance abuse. Antidepressants and anti-anxiety agents should be used carefully. On the one hand, they may profoundly reduce PTSD symptoms such as intrusive recollections of the traumatic experience and physiologic reactivity associated with the disorder. When *appropriately* prescribed, antidepressants may make it easier for the patient to maintain sobriety and avoid self-medication with alcohol. Several studies (citation here) have shown that the anti-anxiety agent, benzodiazepine, might be particularly efficacious in PTSD.

On the other hand, antidepressant medication should not be used for depressive symptoms during the first few weeks following detoxification, since such symptoms often resolve without medication after the patient has been abstinent for several weeks. Anti-anxiety agents, especially benzodiazepines, may need to be particularly avoided due to the risk of addiction and chemical abuse/dependency in susceptible patients. Antabuse can be used successfully as an adjunct in patients with sufficient motivation and impulse control.

Treatment Outcome Research on PTSD

The professional literature on PTSD is increasing, and treatment research is comprising a growing proportion of this literature (Blake et al., 1992). What can the clinician learn from the existing PTSD treatment outcome research? First, behavioral studies appear to be over-represented in the existing treatment outcome research; a paucity of data exists examining psychodynamic and other therapies with PTSD. This imbalance is problematic since it is likely that most PTSD treatments are not primarily behavioral. As a result, the outcome data currently available may not accurately reflect conventional PTSD treatment. It is noteworthy, nevertheless, that some studies have found generic or conventional psychotherapy to not produce significant gain with PTSD patients (Boudewyns & Hyer, 1990; Brooks & Scarano, 1985; Cooper & Glum, 1989; Foa et al., 1991). This section reviews the published PTSD treatment studies, focusing primarily on comparative, group, and controlled single subject research. Pharmacotherapeutic approaches are reviewed elsewhere (Friedman, 1988; van der Kolk, 1987, 1983) and will not be described here. Other psychotherapy procedures have also been reported, such as Eye Movement Desensitization - Revised (Shapiro, 1991); however, due to

space constraints, only the studies that adequately demonstrate experimental control are reviewed.

Eight studies have compared different PTSD treatments. In an early PTSD treatment comparison study, Brooks and Scarano (1985) randomly assigned Vietnam combat veterans to either Transcendental Meditation (TM) or psychotherapy (generic) treatment conditions. After three months of weekly treatment, the TM subjects, but not the psychotherapy subjects, showed significant improvement in PTSD and related symptoms. More recently, Frank et al. (1988) compared cognitive therapy to systematic desensitization in treating female rape victims. Both treatments lead to reduced anxiety, depression, and fear, with no statistically significant difference between the two. Resick et al. (1988) compared assertion training, stress inoculation, and supportive psychotherapy plus information/education in the treatment of acutely-distressed rape victims. All three treatments lead to significant improvements in anxiety and depression, but no treatment was superior to any other.

The remaining five outcome studies include comparisons with imaginal flooding. Cooper and Clum (1989) randomly assigned Vietnam combat veterans (n = 14) to either "conventional" PTSD treatment or this same treatment plus imaginal flooding. Only the flooded veterans showed significant symptom reduction. Boudewyns and Hyer (1990) randomly assigned combat veteran PTSD inpatients (n = 38) to receive imaginal flooding or individualized counseling. The inpatients in the flooding condition, but not the counseling condition, showed decreased heart rate when exposed to combat stimuli. In addition, the flooded patients showed significant gains on a composite symptom index at three-months follow-up. Boudewyns et al. (1990) found that a significant proportion of their most successful PTSD inpatients had received flooding (10 of 15), whereas their least successful inpatients largely had received standard treatment (12 of 15).

Brom, Kleber, & Defares (1989) compared desensitization treatment, hypnotherapy, psychodynamic therapy, and a wait-list control condition in the treatment of 112 individuals diagnosed with PTSD. The PTSD patients (primarily crime victims and vehicle accident survivors) were randomly assigned to a treatment and, while improvements were seen in all but the control condition, no differences were found among the treatments. Foa et al.(5) compared stress inoculation therapy (SIT), prolonged exposure (PE; i.e., imaginal exposure), supportive counseling (SC), and wait-list control (WL) conditions in the randomly-assigned treatment of 45 female rape victims diagnosed with PTSD. On measures of PTSD, anxiety, and depression, SIT, followed by PE, were found to be superior immediately following treatment; PE, followed by SIT, were superior at 4 month follow-up.

Three group studies have compared PTSD treatments to no-treatment. Peniston (1986) employed EMG biofeedback-assisted systematic desensitization procedure with eight traumatized Vietnam combat veterans. As compared to controls, the treated veterans showed reduced forehead tension and decreased nightmares and flashbacks. These effects were maintained across a two-year follow-up. Keane et al. (1989) provided implosive therapy to 11 Vietnam

veterans with PTSD, as compared to 13 veterans who did not receive implosive therapy. Implosive therapy was found to reduce PTSD symptoms, depression, impulsivity, guilt, and fear. However, no gains were observed in the affective numbing and avoidance aspects of the veterans' PTSD. Resick and Schnicke (1992) examined Cognitive Processing Therapy (CPT) for 19 female rape victims, as compared to 20 victims who did not receive CPT. Only the women who received CPT showed significant reductions in PTSD and depression.

Most of the controlled single-subject studies on PTSD treatment has examined implosive therapy (imaginal flooding) administered to traumatized combat veterans (Black & Keane, 1982; Fairbank, Gross, & Keane, 1983; Fairbank & Keane, 1982; Keane & Kaloupek, 1982; Mueser, Yarnold & Foy, 1991). All of these studies have shown that flooding can dramatically reduce distress, intrusive memories, and physical arousal. Similarly, Saigh(1987a; 1987b; 1986) demonstrated the effectiveness of flooding with war-traumatized children and adolescents. In these studies, significant reductions were observed in fear, academic and behavioral problems, nightmares, intrusive memories, avoidant behavior, and depression. Collectively, these studies provide a compelling case for the utility of exposure-based methods with PTSD.

As a disorder, PTSD has a relatively heterogeneous phenomenology. Individuals who differ in PTSD presentation may respond differently to treatment. The treatments reviewed here appear to be fairly uniform in reducing at least part of the complex of symptoms known as PTSD. Treatment effects are typically seen in reduced "positive" symptoms, i.e., psychophysiological arousal and startle, intrusive thoughts, nightmares, and anger. On the other hand, it is not clear whether these treatments also reduce "negative" symptoms of PTSD, i.e., numbing, alienation, and restricted affect. Implosive therapy, for example, may not be appropriate for treating victims of sexual assault (for a discussion, see Kilpatrick & Best (1984). Similarly, there is growing evidence that not all traumatized Vietnam veterans respond well to implosive therapy (Mueser, & Butler, 1987; Pitman et al., 1991). It is possible that procedures that contain a more graduated therapeutic exposure, such as systematic desensitization, may be preferable. Decision guidelines for the use of exposure based therapy have been enumerated by Litz, Blake, Gerardi, & Keane (1990).

Certain PTSD treatments may be well-suited to one individual but not another and a careful consideration of individual risk factors may be critical. It may be that these treatments, which are predominately behavioral, are effective primarily in suppressing the more easily observed PTSD symptoms and that adjunctive, alternative treatments may be necessary for adequately treating the less evident aspects of PTSD. Perhaps it is with these negative PTSD symptoms that insight-oriented psychodynamic therapy produces its greatest benefit.

The National Vietnam Veterans Readjustment Study

The National Vietnam Veterans Readjustment Study (NVVRS) is the most rigorous and comprehensive study to date of PTSD and other psychological problems in readjusting to civilian life among Vietnam veterans. In its Executive Summary, the following key findings were noted:

* 15.2 percent of all male Vietnam theater veterans currently have PTSD. This represents about 479,000 of the estimated 3.14 million men who served in the Vietnam theater.

* 8.5 percent of all female Vietnam theater veterans currently have PTSD. This represents about 610 of the 7200 women who served.

* An additional 11.1 percent of male theater veterans and 7.8 percent of female theater veterans--350,000 additional men and women, currently suffer from "partial PTSD." That is, they have clinically significant stress reaction symptoms of insufficient intensity or breadth to qualify as full PTSD, but may still warrant professional attention.

* NVVRS analyses of the lifetime prevalence of PTSD indicate that nearly one-third (30.6 percent) of male Vietnam theater veterans (over 960,000 men) and over one fourth (26.9 percent) of women serving in the Vietnam theater (over 1900 women) have had the full-blown disorder at some point since returning from Vietnam. Thus, about one-half of the men and one-third of the women who have ever had PTSD still have it today. These findings are consistent with the conceptualization of PTSD as a chronic, rather than acute, disorder.

* NVVRS findings indicate a strong relationship between PTSD and other postwar readjustment problems in virtually every domain of life.

* The prevalence of PTSD and other postwar psychological problems is significantly higher among those with high levels of exposure to combat and other war zone stressors in Vietnam, either by comparison with their Vietnam era veteran and civilian peers or with other veterans who served in the Vietnam theater and were exposed to low or moderate levels of war zone stress.

* Substantial differences in current PTSD prevalence rates were also found by minority status. Analyses of several factors that may account for these differences may be attributed to the differing levels of exposure to war zone stress and/or that minority veterans experienced more mental health and life adjustment problems subsequent to their service in Vietnam than non-minority veterans.

* PTSD has a substantial negative impact not only on the veterans' lives, but also on the lives of spouses, children, and others living with the veterans.

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BIOLOGICAL PERSPECTIVE OF PTSD

Current findings in biological research suggest that patients with PTSD display marked abnormalities in sympathetic nervous system arousal, in hypothalamic-pituitary-adrenocortical function, in the endogenous opioid system, and in the physiology of sleep and dreaming. This information can help us expand our theoretical understanding of PTSD from a purely psychological context to a biopsychosocial model in which many different factors contribute to the pathology of PTSD.

The development of a biological perspective on this disorder can complement psychological diagnostic techniques. The integration of psychosocial and biological assessment can achieve greater precision in identifying PTSD, and in distinguishing PTSD from either major depressive disorder or panic disorder since PTSD exhibits many of the same symptoms of these other psychiatric illnesses.

Historical Overview: Biological Research in Combat Stress

Biological research in the area of combat-related stress is not new. Abraham Kardiner conducted research on World War I veterans during the 1940's (Kardiner, 1941; Kardiner and Spiegel, 1947). Theorizing from the psychological perspective of stress and adaptation, Kardiner stated that (what is now called) PTSD was a physioneurosis in which the patient's adaptive capacity was "smashed."

Later on, Cohen investigated neurocirculatory asthenia (NCA) on a clinical population consisting mostly of combat veterans (Cohen et al, 1948; Cohen and White, 1950). Cohen and associates' research showed that combat veterans with NCA had many abnormalities that could be readily detected when they were asked to perform muscular work on a treadmill. Patients with NCA (a) could not work as long as controls, (b) had a metabolic defect evidenced by less efficient oxygen consumption and higher blood lactate concentration, (c) had reduced pulmonary ventilatory efficiency, (d) had excessively high pulse rates during work, and (e) showed abnormal reactivity to painful stimuli. Placing their work in a historical context of physiological and medical research on combat survivors dating back to the Civil War, they recognized that NCA previously had many other names, including soldier's heart, anxiety neurosis, nervous exhaustion, DaCosta's Syndrome, irritable heart, and effort syndrome. Cohen and his associates speculated about the possible impact of military experience on their experimental observations, and noted that many of their subjects had been in good health prior to the onset of NCA. They also noted that the vast majority of their subjects blamed the army for their difficulties, and that a high percentage of patients reported a harrowing experience in combat. Cohen never went beyond such preliminary observations to explore the possibility that exposure to trauma was the common denominator for many of their subjects and concluded that the cause of NCA was unknown.

Though Cohen's work has recently been "rediscovered" by researchers in the field of panic disorder, recent findings of PTSD patients by biologically oriented investigators suggest that his work is quite relevant to PTSD research as well. For example, Israeli combat veterans with PTSD exhibit low effort tolerance and decreased cardiac reserve in comparison to controls (Shalev et al., 1990). Burn patients with PTSD have significantly lower pain thresholds than burn patients without PTSD (Perry et al., 1987). Reports of a possible link between chronic pain and PTSD (Benedikt and Kolb, 1986; Rapaport, 1987) are also consistent with these observations of hyperalgesia among PTSD patients. Finally, reports of higher rates of somatic complaints among Israeli combat veterans with PTSD (Solomon and Mikulincer, 1987) update similar observations by Cohen et al (1948) among World War II veterans with NCA.

These clinical observations indicate how exposure to trauma may alter the body's normal physiology and health and suggest that PTSD is associated with a number of biological alterations that may be expressed somatically as well as psychiatrically. These are shown in Tables 8 and 9.

TABLE 8

PHYSIOLOGICAL ALTERATIONS ASSOCIATED WITH PTSD

1. Sympathetic nervous system arousal
   a. Elevated baseline sympathetic indices
   b. Excessive response to neutral stimuli
   c. Excessive response to traumamimetic stimuli

2. Excessive startle reflex
   a. Lowered threshold
   b. Increased amplitude

3. A reducer pattern of cortical evoked potentials in response to neutral stimuli

4. Abnormalities in sleep physiology
   a. Increased sleep latency; increased movements; increased awakenings
   b. Decreased sleep time
   c. Possible disturbances in sleep architecture
   d. Traumatic nightmares differ from other types of nightmares

Psychobiological and Pharmacological Approaches to PTSD

PTSD appears to be associated with a complex array of abnormalities in several biological systems. Despite the fact that systematic research in this area is at a relatively early stage, robust findings from a number of experimental approaches suggest that PTSD patients exhibit distinctive physiological, neuropharmacological and neuroendocrinological alterations. In addition, there is a wealth of psychological and neurobiological theory and

data that may be directly applicable to our understanding of PTSD. Moreover, unlike most other psychiatric disorders, there are several animal models that may be directly applicable to PTSD including conditioned fear, (Kolb, 1987, 1988) two factor theory, (Keane et al., 1985) learned helplessness to inescapable shock (van der Kolk, 1985) and kindling (Friedman, 1988, 1991).

In our opinion, psychobiological laboratory techniques designed to elucidate the pathophysiology of this disorder will also lead to the development of clinically useful biological approaches to the diagnosis and treatment of PTSD. Although diagnostic and methodological considerations must be factored into any interpretation of such data, current biological research findings have fostered a growing conviction among many clinicians that pharmocotherapy is sometimes useful in reversing biological abnormalities associated with PTSD.

This review will begin by describing the physiological, neuropharmacological and neuroendocrine abnormalities that current research suggests are associated with PTSD. This should provide a background for understanding and predicting why certain pharmacological approaches might be more effective than others in treating this disorder.

Physiological Alterations in PTSD

As shown in Table 8, physiological findings with PTSD patients (primarily male Vietnam combat veterans) suggest that the homeostat for both the central and autonomic nervous systems has been set at a level of higher arousal. Pulse rate and blood pressure appear to be elevated in the resting state and PTSD patients exhibit greater cardiovascular arousal following exposure to either a neutral stimulus (white noise) or to a meaningful traumimetic stimulus such as the sounds or images of combat (Blanchard and Kolb, 1982; Kolb, 1987; Malloy et al., 1983; Pitman et al., 1987).

The startle response to neutral stimuli in both children and adults indicates that PTSD patients, in contrast to appropriate controls, exhibit both a lower startle threshold as well as significant enhancement of the startle response itself (Ornitz and Pynoos, 1989; Paige et al., 1989).

Paige, et al (1989) report robust differences between PTSD patients and others with respect to lower response thresholds as well as to the pattern of cortical evoked potentials recorded in response to a stimulus pulse of white noise. The pattern of cortical evoked potentials showed a reducer rather than augmenter electrical pattern (Buschbasum, 1976). Paige, et al. suggest that PTSD patients are "reducers" in whom inhibitory feedback loops are activated to dampen a tonic state of hyperarousal.

Finally, sleep and dreaming are altered in PTSD. PTSD patients have difficulty initiating and maintaining sleep. Several studies indicate marked disruption of sleep architecture in PTSD exemplified by increased Stage 1 and Stage 2 sleep, decreased Delta sleep, increased REM latency and decreased total REM percent (Lavie et al., 1979; Kramer et al., 1985; Schlossberg and Benjamin, 1978; Friedman, 1988). Other studies have failed to find such abnormalities

(Greenberg et al., 1972; Kauffman et al., 1987; von Kammen et al., 1987; Ross et al., 1989) and this controversy may actually reflect failure to distinguish PTSD from depressive sleep pathology (see below). Traumatic nightmares are unique phenomena that differ from classic nightmare/night terror Stage 4 episodes as well as from the dream anxiety attacks associated with REM sleep (Friedman, 1981; Ross, 1989). On the one hand, the PTSD dream process is similar to a REM event. Like typical REM events, they appear to the dreamer like a videotape replay sequence. However, since REM sleep is associated with atonia, the nocturnal movements and panic attacks that often accompany traumatic nightmares are more similar to a Stage 4 nightmare/night terror attack.

In short, most physiological data support, DSMIII-R and indicate that PTSD symptoms include hyperarousal, insomnia, and startle. If replicated, the results showing reduction of cortical evoked potentials may actually represent a pathophysiologic aspect of the avoidant/numbing behaviors listed among the PTSD Category C symptoms.

Neurohumoral/Neuroendocrinological Alterations in PTSD

Some, but not all research findings to date indicate that PTSD may be associated with a hyperadrenergic state, abnormal functioning of the hypothalamic-pituitary-adrenocortical (HPA) axis and dysregulation of the endogenous opioid system. These findings are summarized in Table 9.

Pharmacological evidence for increased catecholamines is, of course, consistent with sympathetic hyperarousal noted in Table 8. Twenty-four hour urinary epinephrine and norepinephrine levels in PTSD patients are significantly higher than those of normals and of patients with most other psychiatric disorders (Mason et al., 1986; Losten et al., 1987). Furthermore, PTSD patients have the highest 24-hour urinary norepinephrine/free cortisol ratio of any psychiatric diagnostic group tested to date (Mason et al., 1988). If, as implied by this research, PTSD is associated with higher levels of circulating catecholamines, such increased adrenergic activity should desensitize or down-regulate adrenergic receptors. This, indeed, appears to be the case since the number of both alpha-2 and beta adrenergic receptor sites are reduced in platelets and lymphocytes of combat veterans with PTSD (Perry et al., 1987; Lerer et al., 1987).

TABLE 9

NEUROHUMORAL/NEUROENDOCRINOLOGICAL ABNORMALITIES ASSOCIATED WITH PTSD

1. Increased noradrenergic activity
   a. Increased urinary catecholamine levels
   b. Down-regulation of alpha-2 and beta receptors (reduced platelet MAO activity)
   c. Yohimbine-induced panic and PTSD flashbacks

2. HPA axis abnormalities
   a. Decreased urinary cortisol levels
   b. Elevated urinary catecholamine/cortisol ratio
   c. Increased sensitivity to dexamethasone suppression
   d. Blunted ACTH response to CRH

3. Opioid system dysregulation
   a. Decreased pain threshold at rest
   b. Stress-induced analgesia
   c. Decreased endorphin levels

Additional evidence for CNS adrenergic dysregulation comes from preliminary experiments with yohimbine, an adrenergic alpha-2 antagonist that increases CNS sympathetic arousal by disinhibiting locus coruleus activity. It is well established that yohimbine can precipitate panic attacks in panic disordered patients (Charney et al., 1987). Preliminary experiments with yohimbine at the National Center for PTSD (Southwick et al., 1989) indicate that after Vietnam veterans with PTSD take oral doses of this drug they respond with hyperarousal, anxiety, panic, and intrusive recollections of traumatic combat experiences. In some patients, yohimbine appeared to elicit frank flashback (dissociative) episodes. If these results can be replicated, the fact that such a specific pharmacological probe can precipitate such specific trauma-related symptoms strongly implicates the central adrenergic system in the pathophysiology of PTSD.

HPA abnormalities have also been shown in combat veterans with PTSD. Urinary free cortisol levels are significantly lower among PTSD patients than among other psychiatric diagnostic groups (Mason et al.,1986). There are also preliminary reports (Yehuda et al., 1991) of increased (supersensitive) glucocorticoid receptor activity in the lymphocytes of PTSD patients. This would explain the finding that HPA suppression can be produced in PTSD with very low doses of dexamethasone (0.5mg.) in contrast to normal subjects who require 2 mg. and in contrast to depressed patients who often show no

suppression from 2 mg. Furthermore, PTSD patients exhibit a blunted ACTH (adrenocorticotropin hormone) response to CRH (corticotropin-releasing hormone) in contrast to normal controls (Smith et al., 1989). CRH is the hypothalamic hormone that stimulates the release of ACTH from the pituitary gland. These findings suggest that PTSD is associated with HPA axis abnormalities.

Reports of lower pain thresholds (Perry et al., 1987) and increased susceptibility to chronic pain (Benedikt and Kolb, 1986; Rappaport, 1987) among PTSD patients suggest that this disorder is associated with lower available levels of endogenous opioids. Consistent with this possibility is a finding by Hoffman, et al (1989) who found lower beta-endorphin levels among combat veterans with PTSD. Paradoxically, although opioid levels appear to be reduced in the resting state, the pain threshold may become elevated after exposure to traumamimetic stimuli. Pitman and associates (1988) exposed Vietnam veterans with PTSD to combat scenes from the movie *Platoon*. They found that pain thresholds increased significantly after such exposure. This response, which they believe is a clinical example of what psychologists call stress induced analgesia (SIA), results from a sudden increase in opioid levels following exposure to stressful stimulation. Moreover, Pitman and co-workers were able to prevent the development of SIA by pretreatment with the narcotic antagonist, naloxone, indicating that SIA mediated by the endogenous opioid system is dysregulated by PTSD. van de Kolk, et al (1989) suggest that endogenous opioids are released to attenuate the extreme arousal triggered by the traumamimetic stimuli and they have shown that PTSD patients exposed to such stimuli exhibit greater anxiety, anger, guilt, and dysphoria following injection of the antagonist, naloxone, than following a placebo.

Finally, since the adrenergic, HPA, and opioid systems are related to one another through a variety of neuropharmacological feedback loops, and since many experimental analogs to PTSD abnormalities can be reproduced in laboratory animals exposed to inescapable shock, there is growing confidence in some circles that the complex puzzle of PTSD's pathophysiology will be solved by further experimentation.

Neurobiological Models of PTSD

A number of models have been proposed to integrate the biological abnormalities and clinical symptoms associated with PTSD. All of these models presuppose hyperarousal of the central noradrenergic system and focus especially on the locus ceruleus because it is instrumental in the neurobiology of arousal and panic.

Kolb (1987) has postulated that the excessive and prolonged high intensity stimulation from traumatic exposure produces cortical neuronal and synaptic changes in patients with chronic PTSD. Kolb hypothesizes that the conditioned fear response of PTSD is associated with alteration in brain functions that control aggressive expression and the sleep-dream cycle. This model accounts for the dramatic psychophysiological response in PTSD patients following exposure to traumagenic stimuli. van der Kolk and

associates (1985) have proposed that the animal model of learned helplessness to inescapable shock may be directly applicable to PTSD. They hypothesize that long-term potentiation of locus ceruleus pathways to the hippocampus and amygdala may produce the hyperarousal, traumatic nightmares, and flashbacks that characterize PTSD. The inescapable shock theory offers a neurobiological rationale for stress induced analgesia and for the "action junkie" behavior that is sometimes considered secondary to PTSD.

TABLE 10

BIOLOGICAL DIAGNOSTIC TESTS FOR PTSD

	PTSD	MDD	PD
I. Psychophysiological Responses to Traumagenic Stimuli			
Sympathetic arousal	+	-	-
Stress induced analgesia	+	-	-
II. HPA Axis Abnormalities			
DST	-	+	-
Urinary Cortisol	d	i	?
III. Sodium Lactate Infusion			
Panic Attacks	?	-	+
Flashbacks	?	-	-
IV. Sodium Amytal Interview	+	-	-
V. Sleep EEG			
Initiation and maintenance	d	d	d
Movements during sleep	i	o	i
Sleep architecture	?	+	-

* + ~ proven diagnostic value, - ~ no apparent diagnostic value, ? ~ unknown diagnostic value, d ~ reduced, i ~ increased

van der Kolk (1987) and Friedman (1988) have independently suggested that kindling is a neurobiological model that may be as applicable to PTSD as it is to a cocaine model of psychosis. Kindling is a process by which neuroanatomic structures, especially those in the limbic system, become increasingly sensitized following repeated exposure to electrical stimulation or cocaine-like drugs. Kindling can lead progressively to profound neurophysiological abnormalities such as grand mal seizures or the progressive development of aberrant behavior. This model also suggests that an anti-kindling drug such as carbamazepine might be pharmacologically efficacious in PTSD.

Biological diagnostic tests for PTSD that may be useful for diagnosing PTSD include psychophysiological assessment, the dexamethasone suppression test (DST), sodium lactate infusion and the sodium amytal interview. Many of these same tests have also been used in the diagnosis of Major Depressive Disorder (MDD) and Panic Disorder (PD). As such they have potential for distinguishing PTSD from these other disorders.

Psychophysiological Assessment

At present, psychophysiological assessment is the best and most specific biological diagnostic test for PTSD. This diagnostic technique is based on the fact that traumagenic stimuli elicit sympathetic hyperarousal. The technique is both sensitive and powerful when one uses a general stimulus such as an audiotape of combat sounds or a visual excerpt from a movie such as *Platoon*. It has been found to be even more discriminatory when the provocative stimulus is an individualized autobiographical traumatic anecdote (Pitman et al., 1987). It is important to remember that in the development of standard diagnostic approaches, biological markers in PTSD patients should be assessed both at baseline and immediately after provocation by traumagenic stimuli.

Dexamethasone Suppression Test

The dexamethasone suppression test is widely use in diagnosing major depressive disorder (MDD), and initial research findings indicated the DST could be useful in distinguishing PTSD from MDD (Friedman, 1988; Kudler et al., 1987). These researchers found that patients with PTSD tended to have normal DST's and could therefore be classified as suppressors, whereas patients with both PTSD and MDD were non-suppressors. However, further research has demonstrated that patients with co-diagnosis of MDD+PTSD tended to be suppressors and patients with MDD were non-suppressors (Halbreich et al., 1988). These finding suggest that 1) the DST may have limited value in distinguishing PTSD from MDD and 2) when both MDD and PTSD occur simultaneously, each may alter the biological expression of the other. Very recent work by Yehuda et al. (1991) indicated that PTSD patients may be "supersuppressors" who respond to 0.5 mg. dexamethasone in contrast to normals who require 2.0 mg dexamethasone and in contrast to MDD patients who are often suppressors.

Sodium Lactate (or Yohimbine) Infusion

One of the most definitive tests for Panic Disorder (PD) is the sodium lactate infusion. PD and PTSD share many characteristics in common. Both disorders may be associated with locus ceruleus dysregulation since both exhibit sympathetic hyperarousal and sudden surges of anxiety. In addition, PTSD flashbacks may meet DSMIII-R diagnostic criteria for panic attacks. For these reasons it would be very interesting to learn whether sodium lactate can induce PTSD symptoms as it can panic attacks. Research findings in this area have been inconclusive but it is an area that continues to be investigated, along with other provocative tests for PD such as carbon dioxide inhalation and yohimbine challenge.

Sodium Amytal Interview

PTSD has revived interest in narcosynthetic exploration of repressed traumatic experiences or dissociative episodes triggered by traumagenic stimuli. After decades of neglect, the sodium amytal interview is proving to be a useful clinical tool for identifying catastrophic stressors that are too terrifying for discussion in the normal state of consciousness. Clearly, this diagnostic technique has a unique applicability to PTSD in contrast to MDD or PD.

The amytal interview is not an end in itself but rather a technique for exposing material through narcosynthetic abreaction that must be worked through in subsequent psychotherapy. Candidates for this approach are individuals who have complete or partial amnesia or recurrent episodes of abnormal behavior in which they may become aggressively threatening or violent. Such dissociative episodes are often precipitated by traumagenic stimuli and seem more likely to occur if the patient has been drinking beforehand. The key to the narcosynthetic approach is videotaping the entire session. After full recovery of consciousness, the patient reviews the entire tape with his or her therapist so that recently repressed information can now be incorporated into ongoing psychotherapy. A review of the work of Lawrence Kolb (1985) will give interested clinicians further details on indications, contraindications, and the specific technique for the amytal interview.

Sleep EEG

Current research in the use of the sleep EEG in differentiating PTSD and MDD and PD has resulted in mixed findings. As indicated earlier, there is a controversial literature on possible abnormalities in the sleep architecture of PTSD patients. Less controversial is the uniqueness of the traumatic nightmare which is neither a REM dream anxiety attack nor a non-REM night terror/nightmare episode. The sleep EEG should easily distinguish PTSD from PD, since there is apparently no disturbance of the sleep architecture in PD as there is in PTSD. The discrimination between MDD and PTSD is more difficult to ascertain. Depressed patients exhibit reduced REM latency, reduced Delta sleep and the duration of the first REM period is prolonged. Whether there are enough differences between MDD sleep patterns and PTSD sleep patterns has not been conclusively determined. Additionally, as mentioned earlier in regard to the DST, the simultaneous occurrence of MDD and PTSD may alter the unique biological expression of each disorder, so any specificity for one disorder could be lost in the interaction of the two.

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CLINICAL PSYCHOPHARMACOLOGY IN THE TREATMENT OF PTSD

Based on our current understanding of the biological alterations associated with PTSD, it appears that any drug that can reduce excessive noradrenergic activity will be beneficial in its treatment. This could be accomplished by the use of beta-adrenergic blocking agents such as propranolol, which diminish sympathetic nervous system arousal, by use of tricyclic antidepressants or clonidine, which reduce locus ceruleus activity, or by drugs such as carbamazepine, which act as anti-kindling agents.

Tricyclic Antidepressants

Clinical reports suggest that tricyclic antidepressants (TCAs) reduce specific PTSD symptoms such as hyperarousal, intrusive recollections, flashbacks, and traumatic nightmares. It is important to understand that although the antidepressant action of TCAs is often useful against depressive symptoms associated with PTSD, the primary therapeutic target symptoms under discussion here are PTSD and not MDD symptoms.

Anecdotal reports and open trials using rating scales have generally reported that TCAs reduce DSM-III-R intrusive recollection and hyperarousal symptoms but have little effect on avoidant symptomatology in combat veterans. Similar results have been obtained with different traumatized cohorts such as accident victims, burn patients, and Cambodian concentration camp survivors. Research on TCAs has resulted in conflicting reports, with some investigators finding that TCAs (specifically imipramine) were most effective on PTSD intrusive symptoms (Franke et al., 1988). Results with amitryptyline (Davidson et al., 1990) were modest while desipramine was without effect (Reist et al., 1989). These conflicting findings support the need for continued research in this area and raise a number of important methodological questions.

MAO Inhibitors

MAO Inhibitors are attractive agents to consider in the treatment of PTSD because of their proven efficacy against the sympathetic dysregulation of panic disorder, their antidepressant activity, and their inhibition of REM sleep. Research with MAO Inhibitors has been limited to phenelzine. The findings of these studies suggest that phenelzine's primary PTSD effect is on intrusive rather than avoidant symptoms, although reduction in general anxiety and depressive symptoms were also prominent. The two controlled trials of phenelzine are contradictory. In one trial, phenelzine was very effective (Franke et al., 1988), while in the other, it was no better than placebo (Shestatsky et al., 1988).

The decision to prescribe phenelzine does have to take into consideration a realistic expectation of patient compliance with regard to dietary restrictions and abstention from alcohol, opiates, and other drugs. High rates of

alcoholism and chemical abuse/dependency in combat veterans with PTSD may therefore preclude extensive use of phenelzine.

Methodological concerns about past investigations of TCAs and MAO Inhibitors include: a) length of the drug trial, b) adequate instruments for monitoring drug efficacy, and c) controlling for the potentially confounding influence of MDD among PTSD patients.

Carbamazepine

Carbamazepine is an anticonvulsant that was introduced into psychiatry in 1976 by Post and Kopanda, who suggested that it be prescribed in lithium-refractory bipolar affective disorder. They based this suggestion on a kindling model of endogenous psychosis. Since there are reasons to hypothesize that kindling may also occur in PTSD, carbamazepine is of interest from both a theoretical and clinical standpoint (van der Kolk, 1987; Friedman, 1988).

To date there are no controlled trials of carbamazepine. Open studies using this drug with PTSD combat veterans have shown marked reductions in intensity and frequency of the intrusive or "reexperiencing" symptoms of PTSD such as recurrent nightmares, flashbacks, and intrusive recollections. Additionally, there has been some success in alleviating impulsivity, violent behavior and angry outbursts (Lipper et al., 1986; Wolf et al., 1988).

Propranolol

Propranolol is an adrenergic beta-blocker that has documented efficacy in the treatment of anxiety and panic disorder. It is an attractive drug to consider because it would be expected to antagonize the peripheral and the central sympathetic hyperarousal associated with PTSD. Another advantage of this drug is that it is a non-benzodiazepine anxiolytic that can be prescribed without fear of fostering addiction or chemical abuse or dependence in susceptible PTSD patients. In an A-B-A design (off-on-off medications) with 11 children with acute PTSD (Famularo et al., 1988) propranolol produced significant reduction in PTSD intrusive and arousal symptoms.

Clonidine

Clonidine is an alpha 2 adrenergic agonist currently used in hypertension and opiate withdrawal. It reduces central adrenergic activity by reducing locus ceruleus activity. For that reason it holds out promise as an effective antidote to the adrenergic hyperactivity associated with anxiety disorders.

Initial research findings in open trials with this drug with Vietnam veterans with PTSD were favorable (Kolb et al., 1984). Patients demonstrated lessened explosiveness, reduced nightmares, improved sleep, and lessened startle, intrusive thinking, and hyperalertness. However, controlled studies are needed to validate the consistency of these responses.

Benzodiazepines

Benzodiazepines are potent anxiolytics that have been prescribed widely for PTSD, despite the lack of proven efficacy in controlled trials. Use of

benzodiazepines in PTSD, of course, carries with it the risk of addiction and chemical abuse/dependency in susceptible patients. Practical clinical concerns about addiction notwithstanding, the kindling model of PTSD indicates that there may be a neurobiological rationale for prescribing these drugs. Several studies have shown that benzodiazepine receptor binding is increased significantly during the development of limbic kindling. This suggests that benzodiazepines and other GABA agonists or synergists might be particularly efficacious in PTSD.

Alprazolam

Alprazolam is a triazolo-benzodiazepine that apparently differs from other benzodiazepines because of its demonstrated antipanic and antidepressant properties. It is currently used widely in PTSD although there presently are no double-blind studies demonstrating its efficacy. In addition to concerns about addiction and dependence mentioned previously with regard to all benzodiazepines, alprazolam's pharmacokinetic properties have raised additional concerns. Specifically, its short half-life makes the risk of rebound anxiety and serious withdrawal symptoms greater for alprazolam than for other benzodiazepines that are eliminated more slowly.

Lithium

Lithium has been suggested as an effective treatment for PTSD even in patients with no personal or family history of bipolar or cyclothymic illness. van der Kolk (1987) found that 14 out of 22 PTSD patients tried on lithium reported markedly diminished autonomic hyperarousal, a decreased tendency to react to stress as if it were a recurrence of their original trauma, and a marked decrease in alcohol intake. He did state however that the therapeutic response to lithium in his patients was "clinically indistinguishable" from results found with carbamazepine.

Neuroleptics

The last drugs to consider are the neuroleptics or antipsychotic agents. In the late 1960's and early 1970's, neuroleptics were often prescribed by psychiatrists impressed by the Vietnam veteran's agitation, bizarre and explosive behavior, rage, anti-authoritarian beliefs merging into paranoia, and brief psychotic episodes that we now call flashbacks. Since that time, we have learned that adrenergic hyperarousal rather than psychotic thinking is the primary target in the pharmacotherapy of PTSD. Reduction of DSM-III-R intrusive recollections and arousal symptoms by TCAs, MAO Inhibitors or other drugs is often sufficient to reduce or eliminate psychotic appearing manifestations of PTSD in most patients.

Generally speaking, neuroleptics have no place in the routine treatment of PTSD. If required, they should be used cautiously as a second or third choice following clinical trials of TCAs or other potential first-line drugs. Indications for neuroleptics include aggressive psychotic symptoms (frequently paranoid), overwhelming anger, fragmented ego boundaries, self-destructive behavior, and

frequent flashback episodes characterized by visual and auditory hallucinations of traumatic events.

Comparative Pharmacotherapy: PTSD, MDD, AND PD

Table 11 shows similarities and differences in the psychopharmacologic spectrum of action for PTSD, MDD, and PD. All three disorders appear to respond to TCAs, MAO Inhibitors and probably to alprazolam. Carbamazepine and lithium, which may be effective in PTSD and MDD, are without potency in PD. Although both drugs are efficacious in bipolar affective disorder, only lithium has proven therapeutic value in MDD. Benzodiazepines and anti-adrenergic agents such as propranolol and clonidine, which may be useful in PTSD and PD, can worsen the symptoms of MDD. Finally, neuroleptics which may be useful in a carefully selected minority of PTSD cases have limited usefulness in psychotic MDD and are of no value in PD.

TABLE 11

RESPONSE TO MEDICATION: PTSD, MDD and PD*

	PTSD	MDD	PD
Tricyclic Antidepressants	+	+	+
MAO Inhibitors	+	+	+
Carbamazepine	(+)	?	0
Lithium	(+)	+	0
Benzodiazepines	(+)	-	+
Alprazolam	(+)	+	+
Propranolol	(+)	-	+
Clonidine	(+)	-	(+)
Neuroleptics	+	+	0

* + = proven therapeutic efficacy, (+) = promising uncontrolled trials, 0 = ineffective, - = worsens condition

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THE NATIONAL CENTER FOR POST-TRAUMATIC STRESS DISORDER

The National Center for Post-Traumatic Stress Disorder was mandated by the U.S. Congress in 1984 under Public Law 98-528 to carry out a broad range of multidisciplinary activities in research, education and training. Such initiatives support system-wide efforts to understand, diagnose, ant treat PTSD in veterans who have developed psychiatric symptoms following exposure to war zone stress. The current National Center for PTSD was established in 1989 following a national competition in response to a Request for Proposals issued by VA Central Office. At this time, the National Center is a seven part consortium with Divisions located at VA Medical Centers in White River Junction, VT, West Haven, CT, Boston, MA, Menlo Park, CA and Honolulu, HI.

Executive and Resource Division
Located at the White River Junction VAMC & ROC in Vermont. Directed by Matthew J. Friedman, MD, PhD, this carries out strategic planning, directs the overall operation of the National Center, and interfaces with VA and non-VA programs and organizations. It houses the PTSD Resource Center, directed by Fred Lerner, D.L.S., which has established PILOTS, a comprehensive bibliographic database of the Published International Literature on Traumatic Stress. This office also publishes the *PTSD Research Quarterly*, a newsletter reviewing recent PTSD literature.

Behavioral Science Division Located at the Boston VAMC, this division is under the direction of Terence Keane, PhD. The division is at the forefront of efforts to develop scientifically validated instruments to measure PTSD. Current investigations address both psychological and psychophysiological assessment procedures. In addition, Division staff conduct research on basic mechanisms of PTSD related to cognitive information processing, family and social support factors, and gender issues. Studies of behavioral treatment process and outcome comprise a third major focus of this division's research. Training activities at Boston emphasize both research and clinical skills.

Clinical Neuroscience Division
Located at the West Haven VAMC, this division is under the direction of Dennis S. Charney, MD. This division studies the effects of severe stress on brain fucntion and develops new treatments for trauma victims. It consists of four separate laboratories specializing in neuropharmacology and neuroendocrin-ology, brain imaging, clinical psychoparmacology, and genetics and family studies. In addition to clinical investigations, much emphasis is placed upon training the next generation of researchers.

Clinical Laboratory & Education Division
Located at the Menlo Park VAMC, this division is under the direction of Fred Gusman, MSW. This division is built around a 106 bed unit with three distinct treatment programs: Specialized Inpatient PTSD Programs, PTSD/Alcohol Program, and the Womens Trauma Recovery. Program (WTRP). Each program serves as a major site for inpatient research protocols, sleep studies, and cross-cultural investigations. Established in 1992, the WTRP is the only inpatient program designed to treat female veterans with PTSD. Educational activities include developing a variety of multimedia educational materials and formats, including audiotapes, manuals, publications, telec o nferences, work-shops, form a l conferences, on-site training curricula, and publication of the *National Center for PTSD Clinical Quarterly.* The division also offers disaster mental health training and consultation.

Women's Health Sciences Division
Located at Boston, VAMC. Under the direction of Jessica Wolfe, PhD, this division has pioneered research on the psychological impact of military service on women veterans. Such initiatives include development of psychological assessment techniques, large scale surveys of female Vietnam veterans, and Operations Desert Storm returnees. There has been a special emphasis on the impact of sexual assault and the effect of PTSD on women's health and medical problems. There is also a major focus on treatment and training.

Pacific Islands Division
Located in Honolulu, Hawaii. Under the direction of Raymond Scurfield, DSW, this division places a major focus on research, education, and training concerning cross-cultural factors affectign the expressionk, assessment, and treatment of PTSD among Pacific Islander and Asian-American ethnocultural groups. It also emphasizes epidemiologic and treatment outcome PTSD research. A third focus is mental health service delivery in remote and isolated geographic areas. Finally, there is collaboration with the Department of Defense concerning the acute and chronic impact of military trauma among active duty personnel.

Although not funded by the National Center, the **Northeast Program Evaluation Center** at West Haven VAMC is programmatically linked with all Divisions. Under the direction of Robert Rosenheck, MD., this division performs ongoing evaluation and monitoring of all VA hospital-based PTSD programs throughout the nation.

For more information about the Department of Veterans' Affairs National Center for Post-Traumatic Stress Disorder, please call 415-493-5000 ext. 7314. Our FTS phone number is 700-463-7314.

DEPARTMENT OF VETERANS AFFAIRS: SERVICES FOR PTSD

In addition to the National Center for Post-Traumatic Stress Disorder, the Department of Veterans Affairs offers a variety of services through its nationwide healthcare system for veterans requiring PTSD treatment. These services include:

* specific medical centers that provide specialized inpatient treatment programs specifically for these veterans.

* PTSD clinical teams (PCT's): outpatient clinical teams that provide specialized outpatient care and consultation/liaison services.

* inpatient and outpatient programs that specialize in the treatment of the veteran suffering from both PTSD and alcoholism.

* inpatient treatment programs that specialize in the treatment of women veterans suffering from warzone-related PTSD

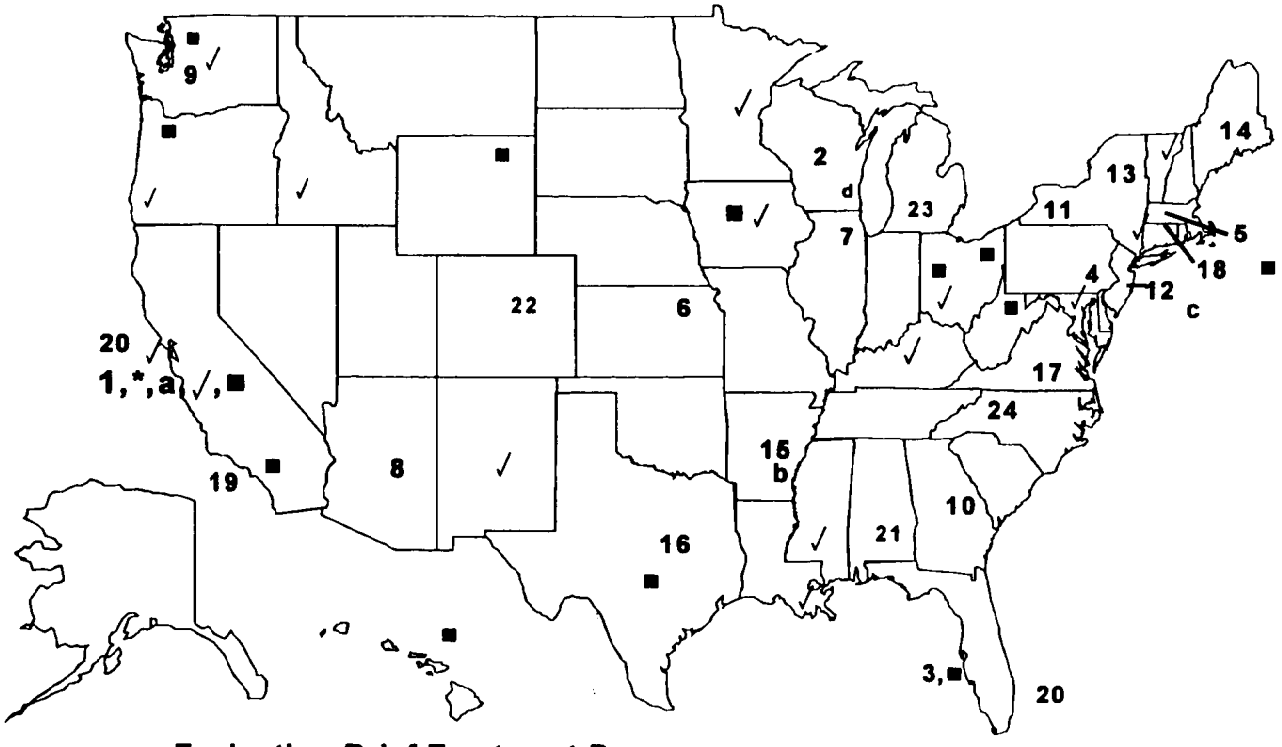
* Readjustment Counseling Service (Vet Center system) which provides various community based services, including counseling and social assistance.

The attached maps and listings identify the locale of each of these services. For more information on these programs, contact your local VA medical center or Vet center.

SPECIALIZED INPATIENT PTSD PROGRAMS
FY 78 - 94

Specialized Inpatient PTSD Units

1. Palo Alto, CA (Fy '78)
2. Tomah, WI (Fy '80)
3. Bay Pines, FL (Fy '81)
4. Coatesville, PA (Fy '82)
5. Northampton, MA (Fy '82)
6. Topeka, KS (Fy '82)
7. North Chicago, IL (Fy '83)
8. Phoenix, AZ (Fy '84)
9. American Lake, WA (Fy '85)
10. Augusta, GA (Fy '85)
11. Buffalo, NY (Fy '85)
12. Lyons, NJ (Fy '85)
13. Montrose, NY (Fy'85)
14. Togus, ME (Fy '86)
15. Little Rock, AR (Fy '87)
16. Waco, TX (Fy '88)
17. Salem, VA (Fy '90)
18. West Haven, CT (Fy '90)
19. West Los Angeles, CA (Fy '90)
20. Miami, FL (Fy '91)
21. Tuskegee, AL (Fy '94)
22. Denver, CO (Fy '94)
23. Battle Creek, MI (Fy '94)
24. Salisbury, NC (Fy '94)



PTSD Residential Rehabilitation Programs

- Martinsburg, WV (Fy '91)
- Sheridan, WY (Fy '91)
- Temple, TX (Fy '91)
- Brecksville, OH (Fy'91)
- Dayton, OH (Fy '92)
- Knoxville,IA (Fy'92)
- West Haven, CT (Fy '92)
- Portland, OR (Fy '92)
- Honolulu, HI (Fy'92)
- West Los Angeles, CA (Fy '94)
- Anchorage, AK (Fy '94)
- Seattle, WA (Fy '94)

Evaluation Brief Treatment Program

- ✓Minneapolis, MN (Fy'90)
- ✓ Cincinnati, OH (Fy '91)
- ✓ Denver, CO (Fy '91)
- ✓ Des Moines, IA (Fy '91)
- ✓ New Orleans, LA (Fy '91)
- ✓ Palo Alto, CA
- ✓ Seattle, WA (Fy '91)
- ✓ Jackson, MS (Fy '92)
- ✓ Louisville,KY (Fy'92)
- ✓ San Francisco, CA (Fy '92)
- ✓ West Haven, CT (Fy '94)
- ✓ Boise, ID (Fy '94)
- ✓ Baltimore, MD (Fy '94)
- ✓ Bronx, NY (Fy '94)
- ✓ Roseburg, OR (Fy '94)
- ✓ White River JCT, VT (Fy '94)

PTSD Substance Use Disorder Units (PSU)

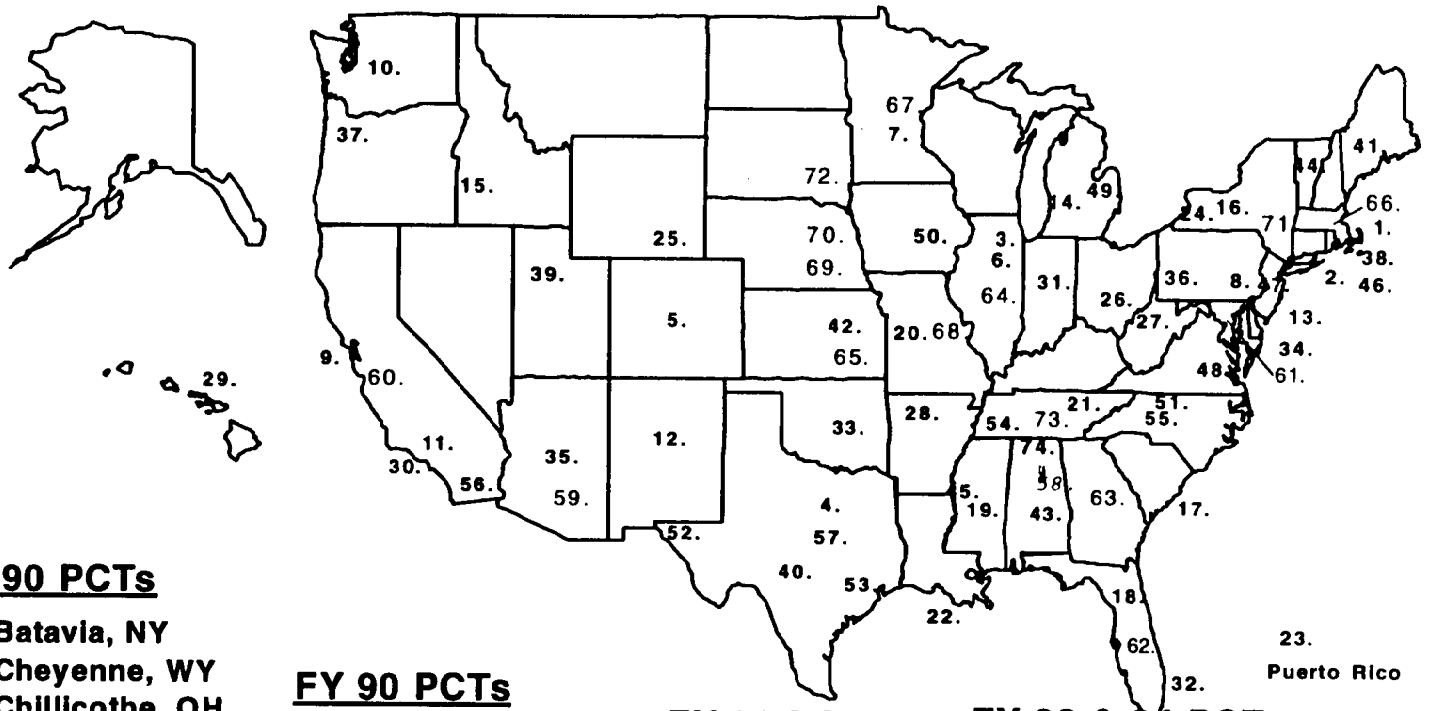
a. Palo Alto, CA (Fy '89)
b. Little Rock, AR (Fy '90)
c. Lyons, NJ (Fy '90)
d. Milwaukee, WI (Fy '90)

Women's Program

* Palo Alto, CA (Fy '91)

December, 1994

Specialized Outpatient PTSD Programs
(Map 1 of 2)



FY 87 PCTs

1. Boston, MA
2. Brooklyn, NY
3. Chicago, IL
4. Dallas, TX
5. Denver, CO
6. Hines, IL
7. Minneapolis, MN
8. Philadelphia, PA
9. San Francisco, CA
10. Seattle, WA
11. Commerce, E. LA, CA

FY 89 PCTs

12. Albuquerque, NM
13. Baltimore, MD
14. Battle Creek, MI
15. Boise, ID
16. Canandaigua, NY
17. Charleston, SC
18. Gainesville, FL
19. Jackson, MS
20. Kansas City, MO
21. Mountain Home, TN
22. New Orleans, LA
23. San Juan, PR

FY 90 PCTs

24. Batavia, NY
25. Cheyenne, WY
26. Chillicothe, OH
27. Clarksburg, WV
28. Fayetteville, AR
29. Honolulu, HI
30. Loma Linda, CA
31. Marion, IN
32. Miami, FL
33. Oklahoma City, OK
34. Perry Point, MD

FY 90 PCTs

35. Phoenix, AZ
36. Pittsburgh, PA
37. Portland, OR
38. Providence, RI
39. Salt Lake City, UT
40. San Antonio, TX
41. Togus, ME
42. Topeka, KS
43. Tuskegee, AL
44. White River JT., VT

FY 91 PCTs

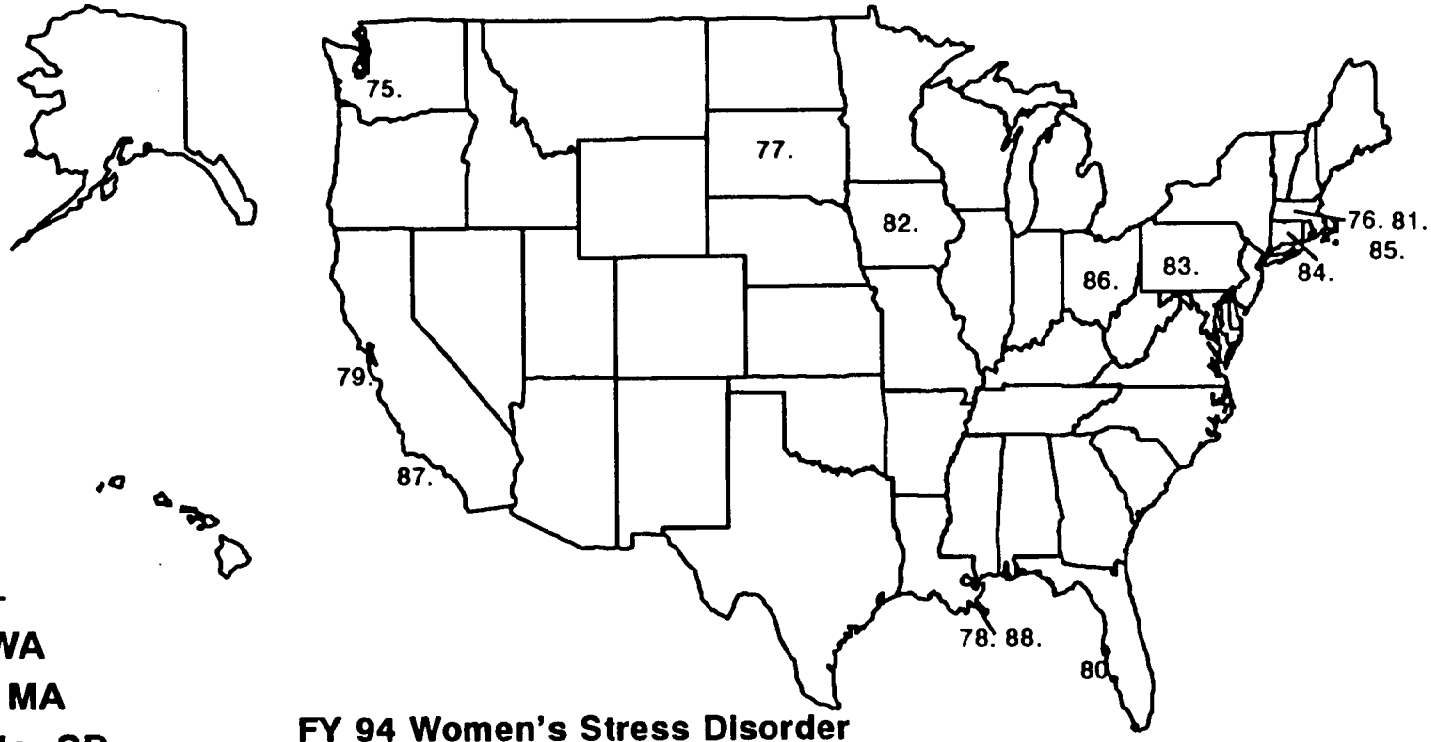
45. Biloxi, MS
46. Bronx, NY
47. Coatesville, PA
48. Hampton, VA
49. Ann Arbor, MI
50. Iowa City, IA
51. Durham, NC
52. El Paso, TX
53. Houston, TX
54. Memphis, TN
55. Salisbury, NC
56. San Diego, CA

FY 92 & 94 PCTs

57. Waco, TX (Fy '92)
58. Tuscaloosa, AL
59. Tucson, AZ
60. Palo Alto, CA
61. Washington, DC
62. Tampa, FL
63. Atlanta, GA
64. Danville, IL
65. Wichita, KS
66. Brockton/West Roxbury, MA
67. St. Cloud, MN
68. St. Louis MO
69. Lincoln, NE
70. Omaha, NE
71. New York, NY
72. Sioux Falls, SD
73. Murfreesboro, TN
74. Birmingham, AL

December 1994

Specialized Outpatient PTSD Programs
(Map 2 of 2)



FY 90 SUPTs

75. Tacoma, WA
76. Brockton, MA
77. Fort Meade, SD
78. New Orleans, LA
79. San Francisco, CA
80. Bay Pines, FL
81. Worcester, MA
82. Knoxville, IA
83. Pittsburgh, PA
84. West Haven, CT

FY 94 Women's Stress Disorder Treatment Teams

85. Boston, MA
86. Brecksville, OH
87. Loma Linda, CA
88. New Orleans, LA